Amendments to the Specification:

Please replace the paragraph beginning on page 6, line 24 with the following amended paragraph:

~~Figure 1 illustrates binding specificities of polyclonal antibodies for p33ING2 and polyclonal antibodies for p33ING1 by ELISA.~~

Please replace the paragraph beginning on page 6, line 26 with the following amended paragraph:

Figure ~~2~~ 1 illustrates binding specificities of polyclonal antibodies for p33ING2 and polyclonal antibodies for p33ING1 by Western blot analysis.

Please replace the paragraph beginning on page 6, line 28 with the following amended paragraph:

~~Figure 3 illustrates that p33ING2 inhibits cell growth of HCT116 cell line by colony formation assay.~~

Please replace the paragraph beginning on page 6, line 30 with the following amended paragraph:

Figure [4] 2 illustrates a Western blot that shows that p33ING2 protein is induced by topoisomerase II inhibitor, etoposide.

Please replace the paragraph beginning on page 6, line 32 with the following amended paragraph:

Figure ~~5~~ 3 illustrates FACScan flow cytometric data that shows that p33ING1 or p33ING2 can induce G_1 cell cycle arrest.

Please replace the paragraph beginning on page 65, line 19 with the following amended paragraph:

~~As shown in Figure 1, by~~ By ELISA (enzyme-linked immunosorbent assay) anti-p33ING2 polyclonal antibodies are reactive with recombinant GST-p33ING2 protein or its peptide fragment KMP-1 (SEQ ID NO:5), but are not cross-reactive with recombinant GST-p33ING1 protein or its peptide fragment KMP-2 (SEQ ID NO:9). Anti-p33ING1 polyclonal antibodies are reactive with recombinant GST-p33ING1 protein or its peptide fragment KMP-2, but are not cross-reactive with recombinant GST-p33ING2 protein or its peptide fragment.

Please replace the paragraph beginning on page 65 line 26 with the following amended paragraph:

As shown in Figure ~~2~~ 1, by Western blot analysis, anti-p33ING2 polyclonal antibodies are reactive with recombinant p33ING2 protein, but are not cross-reactive with recombinant p33ING1 protein. Anti-p33ING1 polyclonal antibodies are reactive with recombinant p33ING1 protein, but are not cross-reactive with recombinant p33ING2 protein.

Please replace the paragraph beginning on page 66, line 5 with the following amended paragraph:

Mammalian expression vectors (with CMV promoter, Neomycin resistant) containing p33ING2 in sense orientation (pcDNA3-ING2) and in antisense orientation (pcDNA3-AntiING2) were constructed. HCT116 cell lines were transfected with the expression vectors. The transfected cells were selected by Neomycin. The colony formation assay was used to test the effect of p33ING2 and anti-p33ING2 expression in HCT116 cell lines. ~~As shown in Figure 3,~~ HCT116 cells transfected with pcDNA3-ING2 formed less colonies compared to HCT116 cells transfected with pcDNA3-AntiING2 or HCT116 cells transfected with pcDNA3 (without any inserts)~~. This result illustrates~~ , demonstrating that p33ING2 inhibits cell growth.

Please replace the paragraph beginning on page 66, line 30 with the following amended paragraph:

Calu6 cells were treated with topoisomerase II inhibitor, etoposide (SIGMA, E-1383, 10 μg/ml). Etoposide can induce DNA damage (e.g., double-strand DNA break). Figure [4] 2 shows the Western blot of p33ING1, p33ING2 and beta-actin (as control). The protein analysis indicated that p33ING2 protein expression was induced by the treatment of Calu6 cells with etoposide. However, p33ING1 protein expression was not induced by etopside.

Please replace the paragraph beginning on page 67, line 5 with the following amended paragraph:

RKO cells were transfected with pcDNA3.1 (control), pcDNA3.1-p33ING1, or pcDNA3.1-p33ING2. Cells were co-transfected with pEGFP-F Amp (a plasmid containing an enhanced green fluorescent protein and an ampicillin transfection marker). The cells were gated by GFP. The GFP-positive cells were considered to be pcDNA3.1, pcDNA3.1-p33ING1, or pcDNA3.1-

p33ING2 positive. The propidium iodide signal was used as a measure for DNA content to determine cell cycle profiles on a FACScan flow cytometer (Becton-Dickinson). *See* Figure ~~5~~ 3. The percentages of the cells in each cell cycle phage were calculated by the ModFit program (Becton-Dickinson), and the results are as follows:

pcDNA3.1 G_0/G_1 (43.1%), G2M (32.5%), S-phase (24.4%)

pcDNA3.1-p33ING1 G_0/ G_1 (67.1%), G_2M (21.7%), S-phase (11.2%)

pcDNA3.1-p33ING2 G_0/ G_1 (71.2%), G_2M (19.9%), S-phase (8.9%)

These results indicate that p33ING1 or p33ING2 can induce G_1 cell cycle arrest in cells.

Amendments to the Drawings:

Original Figures 1 and 3 are cancelled. The attached sheets of drawings includes changes to original Figures 2, 4, and 5. In addition to amendments required by the USPTO draftsman, Figures 2, 4, and 5 are renumbered as Figures 1, 2, and 3 respectively These sheets, which include amended Figures 1, 2 and 3, replace the original sheets including Figures 1-5.

Attachment: Replacement Sheet
Annotated Sheet Showing Changes